FILE 'REGISTRY' ENTERED AT 10:44:44 ON 18 JUN 1999

STRUCTURE FILE UPDATES: 18 JUN 99 HIGHEST RN 225531-94-2
DICTIONARY FILE UPDATES: 18 JUN 99 HIGHEST RN 225531-94-2

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 13, 1999

Please note that search-term pricing does apply when conducting SmartSELECT searches.

=> s 97521-28-3/rn

L2 1 97521-28-3/RN

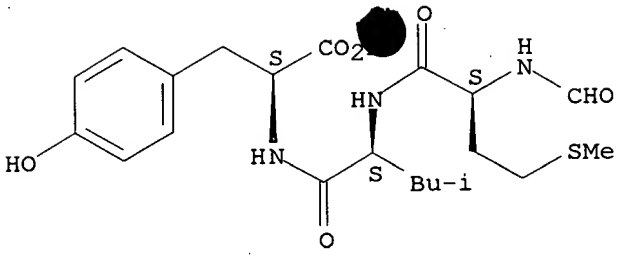
=> d all

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 1999 ACS
RN **97521-28-3** REGISTRY
CN L-Tyrosine, N-formyl-L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Tyrosine, N-[N-(N-formyl-L-methionyl)-L-leucyl]-
OTHER NAMES:
CN N-Formyl-L-methionyl-L-leucyl-L-tyrosine
FS STEREOSEARCH
MF C21 H31 N3 O6 S
CI COM
SR US National Library of Medicine
LC STN Files: BIOSIS, CA, CAPLUS, CHEMCATS, MEDLINE, TOXLINE, TOXLIT

Ring System Data

Elemental Analysis EA	Elemental Sequence ES	Size of the Rings SZ	Ring System Formula RF	Ring Identifier RID	RID Occurrence Count
C6	C6	6	C6	46.150.18	1

Absolute stereochemistry.



6 REFERENCES IN FILE CA (1967 TO DATE)
7 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1

AN 122:312532 CA
TI H-2M3a violates the paradigm for major histocompatibility complex class I peptide binding
AU Vyas, Jatin M.; Rodgers, John R.; Rich, Robert R.
CS Departments Microbiology Immunology, Baylor College Medicine, Houston, TX, 77030, USA
SO J. Exp. Med. (1995), 181(5), 1817-25
CODEN: JEMEAV; ISSN: 0022-1007
DT Journal
LA English
CC 15-2 (Immunochemistry)
AB The major histocompatibility (MHC) class I-b mol. H-2M3a binds and presents N-formylated peptides to cytotoxic T lymphocytes. This requirement potentially places severe constraints on the no. of peptides that M3a can present to the immune system. Consistent with this idea, the M3a-Ld MHC class I chimera is expressed at very low levels on the cell surface, but can be induced significantly by the addn. of specific peptides at 27.degree.. Using this assay, the authors show that M3a binds many very short N-formyl peptides, including N-formyl chemotactic peptides and canonical octapeptides. This observation is in sharp contrast to the paradigmatic size range of peptides of 8-10 amino acids binding to most class I-a mols. and the class I-b mol. Qa-2. Stabilization by fMLF-benzylamide could be detected at peptide concns. as low as 100 nM. While N-formyl peptides as short as two amino acids in length stabilized expression of M3a-Ld, increasing the length of these peptides added to the stability of peptide-MHC complexes as detd. by 27-37.degree. temp. shift expts. The authors propose that relaxation of the length rule may represent a compensatory adaptation to maximize the no. of peptides that can be presented by H-2M3a.
ST H2M3 antigen binding formyl peptide structure
IT Histocompatibility antigens
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(H-2M3a; formyl peptide structure in binding and stabilization of)
IT Molecular structure-biological activity relationship
(histocompatibility antigen H-2M3a-binding; of formylated peptides)
IT Peptides, biological studies
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(N-formyl, structure in binding and stabilization of H-2M3a histocompatibility antigen)
IT 4289-98-9, N-Formylmethionine 15183-28-5, N-Formylmethionylalanine 17351-32-5 22008-60-2, N-Formylmethionylphenylalanine 22029-25-0 29790-45-2 59880-97-6 59881-02-6 59881-03-7, N-Formylmethionylmethionylmethionine 59881-08-2 61864-82-2 65929-03-5 67247-11-4 67247-12-5 70637-32-0 73572-34-6 80180-62-7 80180-63-8 97521-28-3 100929-80-4 127476-71-5 127615-82-1 127615-83-2 134314-56-0 148182-69-8 148182-78-9

148182-81-4 163129-51-9 163133-08-2 163133-09-3 163133-10-6
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(formyl peptide structure in binding and stabilization of H-2M3a histocompatibility antigen)

REFERENCE 2

AN 121:274052 CA
TI Small duct cholangitis induced by N-formyl l-methionine l-leucine l-tyrosine in rats
AU Yamada, Shinji; Ishii, Motoyasu; Liang, Liu Shi; Yamamoto, Takeshi; Toyota, Takayoshi
CS School of Medicine, Tohoku University, Sendai, 980, Japan
SO J. Gastroenterol. (1994), 29(5), 631-6
CODEN: JOGAET
DT Journal
LA English
CC 4-3 (Toxicology)
AB Primary sclerosing cholangitis (PSC) frequently accompanies inflammatory bowel diseases. In an attempt to increase the understanding of the pathogenesis of PSC, the authors studied bile duct changes in rats with colitis which had been given N-formyl L-methionine L-leucine L-tyrosine (fMLT) rectally; fMLT is one of the chemotactic peptides produced by Escherichia coli, and is secreted into the bile by hepatocytes after it enters the portal blood. Transrectal administration of fMLT induced a marked inflammation in the portal triad and mild hepatocyte necrosis on the 4th day. The infiltrating leukocytes in the portal tract were mostly mononuclear cells, which densely infiltrated around the bile ducts. These mononuclear cells appeared to attach to bile duct epithelial cells, and they were more numerous in the smaller bile ducts. Electron microscopy revealed that lymphocytes were in direct contact with bile duct lining cells and that some epithelial cells had degenerated or collapsed. These results suggest that this E. coli-derived peptide may induce cholangitis in the small bile duct through cell-mediated mechanisms. Since these pathol. changes resemble those of the bile duct obsd. in the early stage of PSC, it can be concluded that bacterial chemotactic peptides may play a role in the pathogenesis of small-duct PSC.
ST small duct cholangitis formylmethioninelleucinetyrosine
IT Biliary tract
(bile duct, small duct cholangitis induced by formylmethioninelleucinetyrosine)
IT Biliary tract
(disease, cholangitis, small duct cholangitis induced by formylmethioninelleucinetyrosine)
IT 97521-28-3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(small duct cholangitis induction)

REFERENCE 3

AN 117:106876 CA
TI Isolation and purification of N-formylmethionine aminopeptidase from rat intestine
AU Sherriff, Robert M.; Broom, Murray F.; Chadwick, Vinton S.
CS Dep. Exp. Med., Wellcome Med. Res. Inst., Dunedin, N. Z.
SO Biochim. Biophys. Acta (1992), 1119(3), 275-80
CODEN: BBACAQ; ISSN: 0006-3002
DT Journal
LA English
CC 7-2 (Enzymes)
Section cross-reference(s): 14, 15
AB The intestinal mucosal epithelium is exposed to products of intestinal

bacteria including potent inflammatory N-formylmethionyl oligopeptides. An N-formylmethionine aminopeptidase has been purified 2300-fold from rat intestine and was shown to degrade natural fMet oligopeptides from Escherichia coli culture supernatants with loss of bioactivity (release of specific granule constituents from human polymorphonuclear leukocytes) and immunoreactivity (assessed using a polyclonal anti-fMet-Leu-Phe antiserum). The enzyme (which was specific for N-terminal acylmethionine residues) had a native Mr of 340,000 and comprised four subunits of Mr 82,000. The presence of this enzyme in intestinal mucosa could prevent absorption of intact bioactive fMet peptides produced by commensal bacteria in the gut lumen.

ST formylmethionine aminopeptidase intestine; inflammation formylmethionine peptide bacteria aminopeptidase intestine

IT Inflammation
(formylmethionine aminopeptidase of intestine in relation to)

IT Escherichia coli
(formylmethionine aminopeptidase of intestine reaction with formylmethionine oligopeptides of, inflammation in relation to)

IT Intestine, composition
(formylmethionine aminopeptidase of, purifn. and properties of, inflammatory bacterial peptides in relation to)

IT Kinetics, enzymic
Michaelis constant
(of formylmethionine aminopeptidase, of intestine)

IT Bacteria
(intestinal, formylmethionine aminopeptidase of intestine reaction with formylmethionine oligopeptides of, inflammation in relation to)

IT Peptides, reactions
RL: RCT (Reactant)
(oligo-, 1-(N-formylmethionine)-, reaction of, with formylmethionine aminopeptidase of intestine, kinetics of)

IT 76106-80-4P
RL: PREP (Preparation)
(of intestine, purifn. and properties of, inflammatory peptides of bacteria in relation to)

IT 18321-99-8 22008-60-2 29790-45-2 59880-97-6 60189-52-8
60461-05-4 61864-82-2 73572-34-6 76078-88-1 97521-28-3
RL: RCT (Reactant)
(reaction of, with formylmethionine aminopeptidase of intestine, kinetics of)

REFERENCE 4

AN 111:75641 CA

TI Bacterial chemotactic oligopeptides and the intestinal mucosal barrier

AU Ferry, Dianne M.; Butt, Terence J.; Broom, Murray F.; Hunter, June; Chadwick, Vinton S.

CS Med. Sch., Univ. Otago, Dunedin, N. Z.

SO Gastroenterology (1989), 97(1), 61-7
CODEN: GASTAB; ISSN: 0016-5085

DT Journal

LA English

CC 14-3 (Mammalian Pathological Biochemistry)

AB Intestinal absorption and enterohepatic circulation of N-formyl-Met-Leu-125I-Tyr, a bioactive synthetic analog of the bacterial chemotactic peptide N-formyl-Met-Leu-Phe were investigated in the rat. In ileum and proximal and distal colon, dithiothreitol, which increases mucosal permeability, increased peptide absorption and biliary recovery 4-fold, 70-fold, and 20-fold over control values, resp. When dithiothreitol was combined with d-l-benzyl succinate, a potent inhibitor of intestinal carboxypeptidase, absorption and biliary recovery from ileal loops increased markedly to 40-fold over control, whereas there was no further increase in absorption from colon loops. There was a strong

correlation between biliary N-formyl-Met-Leu-125I-T[redacted] recovery and intestinal absorp[redacted]n of 51Cr-ethylenediaminetetra[redacted]tate, a marker of passive mucosal p[redacted]meability. Thus, in the ileum both enzymic degrdn. and restricted mucosal permeability contribute to the intestinal barrier to luminal bacterial formyl oligopeptides. In the colon, however, enzymic mechanisms are less active and restricted mucosal permeability is the major factor. Abnormalities of the intestinal mucosal barrier to proinflammatory bacterial peptides could play a role in inflammatory disorders of the gut.

ST bacterium chemotactic peptide intestine mucosal barrier

IT Bacteria
(chemotactic oligopeptides of, absorption of, by ileum vs. colon, enzymic degrdn. and mucosal permeability in, inflammation in relation to)

IT Intestine, metabolism
(colon, mucosa, bacterial chemotactic oligopeptides absorption by, enzymic degrdn. and mucosal permeability in, inflammation in relation to)

IT Intestine, metabolism
(ileum, mucosa, bacterial chemotactic oligopeptides absorption by, enzymic degrdn. and mucosal permeability in, inflammation in relation to)

IT Peptides, biological studies
RL: BIOL (Biological study)
(oligo-, bacterial, absorption of, by ileum vs. colon, enzymic degrdn. and mucosal permeability in, inflammation in relation to)

IT 97521-28-3
RL: BIOL (Biological study)
(absorption of, by ileum vs. colon, enzymic degrdn. and mucosal permeability in, inflammation in relation to)

IT 9031-98-5, Carboxypeptidase
RL: BIOL (Biological study)
(in bacterial chemotactic oligopeptide absorption by ileum vs. colon, mucosal permeability in relation to)

REFERENCE 5

AN 108:184554 CA

TI Enterohepatic circulation of bacterial chemotactic peptide in rats with experimental colitis

AU Hobson, Christopher H.; Butt, Terence J.; Ferry, Dianne M.; Hunter, June; Chadwick, Vinton S.; Broom, Murray F.

CS Med. Sch., Univ. Otago, Dunedin, N. Z.

SO Gastroenterology (1988), 94(4), 1006-13
CODEN: GASTAB; ISSN: 0016-5085

DT Journal

LA English

CC 14-7 (Mammalian Pathological Biochemistry)

AB The assocn. of hepatobiliary disorders with colonic inflammation is well recognized. Although the pathophysiol. is obscure, increased permeation of toxic bacterial products across the inflamed gut to the portal circulation might be one mechanism. Potentially toxic metabolites include N-formylated chemotactic peptides that are produced by several species of intestinal bacteria and can be detected in colonic fluid in vivo. To investigate the metabolic fate of one of these low mol. wt. proinflammatory peptides, N-formyl L-methionine L-leucine 125I-L-tyrosine was introduced into colon loops of healthy rats and rats with exptl. colitis induced by rectal instillation of 15% (vol/vol) acetic acid. Biliary excretion of intact peptide over 3 h was 6.4 pmol in normal rats and 49.0 pmol in rats with colitis. Thus, an enterohepatic circulation of synthetic N-formyl L-methionine L-leucine L-tyrosine has been demonstrated in the rat. Exptl. colitis was assocd. with an 8-fold increase in biliary excretion of this proinflammatory bacterial peptide. Proinflammatory

bacterial peptides synthesized by colonic bacteria could be important in the pathophysiol. of colon inflammation and its frequently assocd. hepatobiliary complications.

ST bacteria chemotactic peptide enterohepatic circulation colitis

IT Chemotaxis
(bacterial peptides inducing, enterohepatic circulation of, in colitis)

IT Peptides, biological studies
RL: BIOL (Biological study)
(chemotactic bacterial, enterohepatic circulation of, in colitis)

IT Intestine, disease or disorder
(colitis, bacterial chemotactic peptide enterohepatic circulation in)

IT Circulation
(enterohepatic, of bacterial chemotactic peptide, in colitis)

IT 97521-28-3, N-Formyl L-methionyl L-leucyl L-tyrosine
RL: PROC (Process)
(enterohepatic circulation of, in colitis)

REFERENCE 6

AN 108:53386 CA

TI Uptake of ascorbic acid by leukocytes

AU Moser, Ulrich

CS Dep. Vitam. Nutr. Res., F. Hoffmann-La Roche and Co., Ltd., Basel, CH-4002, Switz.

SO Ann. N. Y. Acad. Sci. (1987), 498(Conf. Vitam. C, 3rd., 1986), 200-15
CODEN: ANYAA9; ISSN: 0077-8923

DT Journal

LA English

CC 13-5 (Mammalian Biochemistry)

AB Ascorbic acid (I) was distributed primarily in the cytosol of guinea pig adrenal glands, in animals fed a normal diet contg. 500 mg I/kg or a marginal diet contg. 50 mg/kg for 4 wk. In porcine adrenal cortical cells, I uptake exhibited satn. kinetics with a Km of 20.5 .mu.M and a Vmax of 7.3 nmol/108 cells. I transport by human granulocytes and mononuclear cells was more complex, consisting of a saturable active transport plus passive diffusion. It was calcd. that at 25-50 .mu.M I, a normal plasma concn., active transport of I contributes 64-72% in granulocytes and 87-89% in mononuclear cells. Glucose inhibits the uptake of I by these cells. I uptake, but not that of glucose, was stimulated up to 3-fold by various N-formyl peptides. Ca2+ plus A 23187 also stimulated the uptake of I. In adrenal cortical cells I transport required Na+, K+, and Ca2+, and was not inhibited by glucose. I uptake by both granulocytes and mononuclear cells was inhibited by D-ascorbic acid and competitively by D-erythorbic acid. Evidently, I uptake into cells with a high I concn. is mediated by a stereospecific active transport mechanism that exhibits different kinetics depending on the cell type examd.

ST ascorbate transport leukocyte

IT Adrenal cortex, metabolism
(ascorbate uptake by, kinetics of)

IT Biological transport
(absorption, of ascorbate by adrenal cortex of lab. animals and leukocytes of humans, kinetics of)

IT Cytoplasm
(cytosol, ascorbate of, of adrenal cortex)

IT Leukocyte
(granulocyte, ascorbate uptake by human, kinetics of)

IT Leukocyte
(mononuclear, ascorbate uptake by human, kinetics of)

IT 50-81-7, Ascorbic acid, biological studies
RL: BIOL (Biological study)
(absorption of, by adrenal cortex of lab. animals and leukocytes of humans, kinetics of)

IT 22008-60-2, N-Formyl-L-methionyl-L-phenylalanine 29790-45-2,

N-Formyl-L-methionyl-L-valine 59880-97-6, N-Formyl-L-methionyl-L-leucyl-L-phenylalanine [redacted]189-52-8, N-Formyl-L-methionyl-[redacted]tryptophan 97521-28-3, N-Formyl-L-methionyl-L-leucyl-L-tyrosine
RL: BIOL (Biological study)
(ascorbate transport by granulocytes of humans response to)

IT 7440-70-2, Calcium, biological studies
RL: BIOL (Biological study)
(ascorbate uptake by adrenal cortex of lab. animals and leukocytes of humans response to)

IT 7440-09-7, Potassium, biological studies 7440-23-5, biological studies
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(ascorbate uptake by adrenal cortex response to)

IT 50-99-7, D-Glucose, biological studies 89-65-6, Erythorbic acid 10504-35-5, D-Ascorbic acid
RL: BIOL (Biological study)
(ascorbate uptake by leukocytes of humans inhibition by)

FILE 'EUROPATFULL' ENTERED AT 09:26:44 ON 22 JUN 1999

=> s l4 or l8

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            15 CHEMOTACTIC/CLM
             1 CHEMOTACTICS/CLM
            16 CHEMOTACTIC/CLM
                 ((CHEMOTACTIC OR CHEMOTACTICS)/CLM)
          1530 PEPTIDE#/CLM
             3 (CHEMOTACTIC PEPTIDE#)/CLM
                 ((CHEMOTACTIC(W)PEPTIDE#)/CLM)
         20140 F/CLM
          3608 MET/CLM
             8 METS/CLM
          3616 MET/CLM
                 ((MET OR METS)/CLM)
           356 LEU/CLM
             2 LEUS/CLM
             3 LEI/CLM
           361 LEU/CLM
                 ((LEU OR LEUS OR LEI)/CLM)
         14691 PHE?/CLM
             0 ((F-MET-LEU-PHE?/CLM))
                 ((F(W)MET(W)LEU(W)PHE?)/CLM)
L9           3 L4 OR L8
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=> d bib, kwic 1-3

L9 ANSWER 1 OF 3 EUROPATFULL COPYRIGHT 1999 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

AN 629617 EUROPATFULL ED 19980510 EW 9818 FS PS
TIEN Heteroatom-bearing ligands and metal complexes thereof.
TIDE Heteroatom-substituierte Liganden und Metallkomplexe davon.
TIFR Ligands contenant des heteroatomes et leurs complexes metalliques.
IN Ramalingam, Kondareddiar, 46 Liberty Drive, Dayton, NJ, US;
Raju, Natarajan, 41 New Road, Kendall Park, NJ, US
PA BRACCO International B.V., 7, De Boelelaan, 1083 HJ Amsterdam, NL
PAN 1771640
AG Chopard, Pierre-Antoine et al, c/o BRACCO Research S.A. 7, Route de Drize, 1227 Carouge, CH
AGN 24952
OS EPB1998021 EP 0629617 B1 980429
SO Wila-EPS-1998-H18-T1
DT Patent
LA Anmeldung in Englisch; Veroeffentlichung in Englisch

DS R AT; R BE; R CH; R DE; R DK; R ES; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL PT; R SE
PIT EPB1 EUROPAEISCHE PATENTSCHRIFT
PI EP 629617 B1 980429
OD 941221
AI EP 94-108968 940610
PRAI US 93-77981 930615
REP EP 179608 A EP 194843 A
EP 544412 A
CLMEN. . . glucose derivatives, fatty acids, substrates for muscarine receptors such as 3-quinuclidinyl benzilate, substrates for dopamine receptors such as spiperone, biotin, **chemotactic peptides**, substrates for benzodiazepine receptors, and hypoxia localizing moieties of structures <image> wherein D is a group of atoms that forms,. . .

L9 ANSWER 2 OF 3 EUROPATFULL COPYRIGHT 1999 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

AN 523356 EUROPATFULL ED 19970307 EW 9651 FS PS
TIEN Burst-test.
TIDE Burst-Test.
TIFR Test de poussee.
IN Nebe, Karl Thomas, Dr.-Med., Gaertnerstrasse 6, W-8400 Regensburg, DE; Hartmann, Karin, Lemaitrestrasse 31, W-6800 Mannheim 31, DE
PA Orpegen Medizinisch-Molekularbiologische Forschungsgesellschaft m.b.H., Czerny-Ring 22, 69115 Heidelberg, DE
PAN 389670
AG Huber, Bernhard, Dipl.-Chem. et al, Patentanwaelte H. Weickmann, Dr. K. Fincke F.A. Weickmann, B. Huber Dr. H. Liska, Dr. J. Prechtel, Dr. B. Boehm Postfach 86 08 20, 81635 Muenchen, DE
AGN 5832
OS EPB1996077 EP 0523356 B1 961218
SO Wila-EPS-1996-H51-T2
DT Patent
LA Anmeldung in Deutsch; Veroeffentlichung in Deutsch
DS R AT; R BE; R CH; R DE; R DK; R ES; R FR; R GB; R GR; R IT; R LI; R LU; R NL; R SE
PIT EPB1 EUROPAEISCHE PATENTSCHRIFT
PI EP 523356 B1 961218
OD 930120
AI EP 92-108864 920526
PRAI DE 91-4117459 910528
REP EP 435226 A
REN HIROSHIMA JOURNAL OF MEDICAL SCIENCES vol. 34, no. 1, March 1985, pages 53 - 60; K.TAGA ET AL.: 'Flow Cytometric Assassment of Neutrophil Oxidative Metabolism in Chronic Granulomatous Disease on Small Quantities of Whole Blood: Heterogeneity in Female Patients.' NATUR WISSENSCHAFTEN vol. 75, 1988, pages 354 - 355; G.ROTHE ET AL.:Dihydrorhodamine 123: a New Flow Cytometric Indicator for Respiratory Burst Activity in Neutrophil Granulocytes." JOURNAL OF IMMUNOLOGICAL METHODS vol. 131, 1990, pages 269 - 275; A. EMMENDOERFFER ET AL.: 'A fast and easy method to determine the production of reactive oxygen intermediates by human and murine phagocytes using dihydrorhodamine 123.' JOURNAL OF LEUKOCYTE BIOLOGY vol. 43, no. 4, April 1988, pages 304 - 310; J.P. ROBINSON ET AL.: 'Measurement of Intracellular Fluorescence of Human Monocytes Relative to Oxidative Metabolism.' ACTA ENDOCRINOLOGICA vol. 124, no. 5, May 1991, pages 589 - 594; G.L.SPADONI ET AL.: 'Enhancement by growth hormone of phorbol diester-stimulated respiratory burst in human polymorphonuclear leukocytes.'
CLMEN 5. Process according to Claim 2, characterised in that a

chemotactic peptide or peptide derivative with an N-terminal formyl-met-residue, especially N-formyl-met-leu-phe, is used as a weak leucocyte stimulant.

L9 ANSWER 3 OF 3 EUROPATFULL COPYRIGHT 1999 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

AN 272929 EUROPATFULL ED 19970108 EW 9612 FS PS
TIEN Enkephalinase for therapeutic use.
TIDE Enkephalinase fuer therapeutische Verwendung.
TIFR Encephalinase pour application theraupeutique.
IN Malfroy-Camine, 2130 Sea Cliff Way, San Bruno California 94066, US;
Nadel, Jay A., 2373 Pacific Avenue, San Francisco California 94044, US;
Borson, Daniel B., 422 Andover Drive, Pacifica California 94044, US
PA GENENTECH, INC., 460 Point San Bruno Boulevard, South San Francisco California 94080, US;
THE REGENTS OF THE UNIVERSITY OF CALIFORNIA, 2199 Addison Street, Berkeley, California 95616, US
PAN 210480; 221077
AG Armitage, Ian Michael et al, MEWBURN ELLIS York House 23 Kingsway, London WC2B 6HP, GB
AGN 27761
OS EPB1996019 EP 0272929 B1 960320
SO Wila-EPS-1996-H12-T1
DT Patent
LA Anmeldung in Englisch; Veroeffentlichung in Englisch
DS R BE; R CH; R DE; R FR; R GB; R LI; R LU; R NL
PIT EPB1 EUROPAEISCHE PATENTSCHRIFT
PI EP 272929 B1 960320
OD 880629
AI EP 87-311366 871223
PRAI US 87-2473 870112
US 87-117779 871105
US 86-946566 861224
REP EP 272928 A
REN BIOCHEMISTRY, vol. 22, 1983, pages 3265-3271, American Chemical Society; J.T. GAFFORD et al.: "Human kidney "Enkephalinase", a neutral metalloendopeptidase that cleaves active peptides" BIOCHEMISTRY, vol. 22, 1983, pages 590-599, American Chemical Society; J. ALMENOFF et al.: "Membrane-bound kidney neutral metalloendopeptidase: Interaction with synthetic substrates, natural peptides, and inhibitors" CHEMICAL ABSTRACTS, vol. 104, no. 7, 17th February 1986, page 246, abstract no. 47819e, Columbus, Ohio, US; L.T. TAM et al.: "The importance of disulfide bridges in human endopeptidase (enkephalinase) after proteolytic cleavage" FED. PROC., vol. 45, no. 3, 1986, page 626, abstract no. 2739; D.B. BORSON et al.: "Enkephalinase inhibitors potentiate tachykinin-induced release of 35SO4- labeled macromolecules from ferret trachea" INT. ARCH. ALLERGY APPL. IMMUN., vol. 82, 1987, pages 372-376; S.C. LAZARUS et al.: "Inflammatory mediators, tachykinins and enkephalinase in airways" PROC. NATL. ACAD. SCI. USA, vol. 82, December 1985, pages 8737-8741; J.C. CONNELLY et al.: "Neutral endopeptidase 24.11 in human neutrophils: Cleavage of chemotactic peptide" ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, vol. 448, 1985, pages 666-668; A.J. TURNER et al.: "Metabolism of cholecystokinin by endopeptidase-24.11" CHEM. PHARM. BULL., vol. 34, no. 1, 1986, pages 275-280; Y. SHIMAMORI et al.: "Specificity of a membrane-bound neutral endopeptidase from rat kidney" NATURE, vol. 316, 29th August 1985, pages 823-826; F. CUTTITTA et al.: "Bombesin-like peptides can function as autocrine growth factors in human small-cell lung cancer" BIOCHEM. J., vol. 233, no. 2, 1984, pages 433-440; R. MATSAS et al.: "The metabolism of neuropeptides. The hydrolysis of peptides, including enkephallins, tachykinins and their analogues, by endopeptidase-24.11" BIOLOGICAL

ABSTRACTS, vol. 81, no. 4, page AB-726, abstract no. 35766, Biological Abstracts, Inc., Philadelphia, PA, US; N.M.A. HOOPER et al.: "The metabolism of neuropeptides: Neurokinin A (substance K) is a substrate for endopeptidase-24.11 but not for peptidyl dipeptidase A (angiotensin-converting enzyme)" CHEMICAL ABSTRACTS, vol. 104, no. 1, 6th January 1986, page 123, abstract no. 1347w, Columbus, Ohio, US; N.M. HOOPER et al.: "Neurokinin B is hydrolyzed by synaptic membranes and by endopetidase-24.11 ('enkephalinase) but not by angiotensin converting enzyme" Goodman and Gilman's: "The Pharmacological Basis of Therapeutics", 7. Ed., 1985, pp. 653-657

CLMEN 13. The use according to claim 5 wherein the endogenous peptide is a

FILE 'USPATFULL' ENTERED AT 09:09:06 ON 22 JUN 1999
L1 26 S (CHEMOTACTIC PEPTIDE#) (5A) (NEUTROPHIL OR MAST)
L2 67083 S 514/?/NCL
L3 4 S L2 AND L1
L4 24 S (CHEMOTACTIC PEPTIDE#)/CLM
L5 7 S L4 AND (INFLAM? OR MAST OR NEUTROPH?)/CLM

=> d bib, kwic 1-7

L5 ANSWER 1 OF 7 USPATFULL
AN 1998:128235 USPATFULL
TI Chemotactic wound healing peptides
IN Postlethwaite, Arnold E., 635 Bethany Rd., Eads, TN, United States 38028
Seyer, Jerome, 1412 Carr Ave., Memphis, TN, United States 38104
Kang, Andrew, 2334 Massey Rd., Memphis, TN, United States 38119
PI US 5824647 19981020
AI US 95-457353 19950601 (8)
RLI Continuation-in-part of Ser. No. US 93-127909, filed on 28 Sep 1993, now patented, Pat. No. US 5436228 which is a continuation-in-part of Ser. No. US 90-626631, filed on 12 Dec 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner: Harle, Jennifer
LREP Scully, Scott, Murphy & Presser
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 1469
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. A **chemotactic peptide** wherein the amino acid sequence of the peptide is selected from the group consisting of SEQ ID NO. 21, SEQ. . .

2. A method for inducing either in vitro or in vivo chemotaxis of fibroblasts and/or **inflammatory** cells comprising administering to the fibroblasts and/or **inflammatory** cells at least one peptide of claim 1 in an amount effective to induce chemotaxis.

3. A method for inducing either in vitro or in vivo proliferation of fibroblasts and/or **inflammatory** cells comprising administering to the fibroblasts and/or **inflammatory** cells at least one peptide of claim 1 in an amount effective in induce cellular proliferation.

6. A **chemotactic peptide** having an amino acid sequence selected from the group consisting of SEQ ID NO. 21, SEQ ID NO. 22, SEQ. . .

7. An analog of **chemotactic peptide** set forth in SEQ ID NO. 21, SEQ ID NO. 22, SEQ ID NO. 23, SEQ ID NO. 24, SEQ. . .

L5 ANSWER 2 OF 7 USPATFULL
AN 1998:111632 USPATFULL
TI Technetium-99m labeled peptides for imaging inflammation
IN Dean, Richard T., Bedford, NH, United States
Lees, Robert S., Brookline, MA, United States
Buttram, Scott, Derry, NH, United States
PA Diatide, Inc., Londonderry, NH, United States (U.S. corporation)
PI US 5807538 19980915
AI US 95-484774 19950607 (8)
RLI Division of Ser. No. US 94-266178, filed on 27 Jun 1994 which is a continuation of Ser. No. US 92-851074, filed on 13 Mar 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Hollinden, Gary E.; Assistant Examiner: Jones, Dameron
LREP McDaniels, Patricia A.; Noonan, Kevin E.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 678
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. A complex formed by reacting a reagent comprising a leukocyte-binding **chemotactic peptide** covalently linked to a radiolabel-binding moiety selected from the group consisting of: Cp(aa)Cp wherein Cp is a protected cysteine and. . . with technetium-99m in the presence of a reducing agent, wherein the complex is capable of accumulating at a site of **inflammation** in a mammalian body.

4. A method for preparing an imaging agent for detecting infection or **inflammation** in a mammalian body comprising the steps of a) providing a reagent comprising a leukocyte-binding **chemotactic peptide** covalently linked to a radiolabel-binding moiety selected from the group consisting of Cp(aa)Cp wherein Cp is a protected cysteine and. . .

6. A method for preparing an imaging agent for detecting infection or **inflammation** in a mammalian body comprising the steps of a) providing a reagent comprising a leukocyte-binding **chemotactic peptide** covalently linked to a radiolabel-binding moiety selected from the group consisting of Cp(aa)Cp wherein Cp is a protected cysteine and. . .

7. A complex formed by reacting technetium-99m with a reagent comprising a leukocyte-binding **chemotactic peptide** covalently linked to a radiolabel-binding moiety, wherein the technetium-99m complexed radiolabel-binding moiety has a net charge of -1, and wherein the complex is capable of accumulating at a site of **inflammation** in a mammalian body.

11. A method for preparing an imaging agent for detecting infection or **inflammation** in a mammalian body comprising the steps of a) providing a reagent comprising a leukocyte-binding **chemotactic peptide** covalently linked to a radiolabel-binding moiety capable of forming a complex with technetium-99m having a net charge of -1; and. . .

12. A complex formed by reacting technetium-99m with a reagent comprising a **chemotactic peptide** which binds to leukocytes covalently linked to a radiolabel-binding moiety, wherein the technetium-99m and the radiolabel-binding moiety form a neutral complex, and wherein the complex is capable of accumulating at a site of **inflammation** in a mammalian body.

L5 ANSWER 3 OF 7 USPATFULL
AN 1998:95223 USPATFULL
TI Labeled chemotactic peptides to image focal sites of infection or inflammation
IN Fischman, Alan J., Boston, MA, United States
Solomon, Howard F., New Hope, PA, United States
Derian, Claudia K., Hatboro, PA, United States
Bridger, Gary J., Bryn Mawr, PA, United States
Higgins, III, John D., West Chester, PA, United States
Larsen, Scott K., West Chester, PA, United States
Hernandez, Pedro E., Malvern, PA, United States
Rubin, Robert H., Brookline, MA, United States
Strauss, H. William, Skillman, NJ, United States
Fuccello, Anthony J., Princeton, NJ, United States
Kroon, Daniel J., Flemington, NJ, United States
PA The General Hospital Corporation, Boston, MA, United States (U.S. corporation)
Johnson Matthey, Inc., West Chester, PA, United States (U.S. corporation)
Ortho Pharmaceutical Corporation, Raritan, NJ, United States (U.S. corporation)
PI US 5792444 19980811
AI US 93-140000 19931022 (8)
RLI Continuation-in-part of Ser. No. US 93-55312, filed on 3 May 1993, now patented, Pat. No. US 5350837 And a continuation-in-part of Ser. No. US 93-56950, filed on 5 May 1993, now abandoned which is a continuation of Ser. No. US 89-349186, filed on 4 May 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Kight, John; Assistant Examiner: Kelley, Lara C.
LREP Sterne, Kessler, Goldstein & Fox P.L.L.C.
CLMN Number of Claims: 73
ECL Exemplary Claim: 1
DRWN 27 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 3217
CLM What is claimed is:

1. A method of detecting a site of infection or **inflammation** in an individual which comprises: a. administering to the individual a diagnostically effective amount of a detectably labeled **chemotactic peptide** X--Y--Leu--Phe--[Z].sub.n --W wherein: X is an amino-protecting group, Y is an amino acid residue, Z is a spacer sequence, n. . . is an intermediary functional group, .nu. is 0 or 1, and M.sup.1 is a diagnostically detectable label; and wherein the **chemotactic peptide** substantially accumulates at the site of infection or **inflammation** and does not substantially accumulate at a site that is not infected or **inflamed**; and b. detecting the **chemotactic peptide**.

2. A method of detecting a site of infection or **inflammation** in an individual which comprises: a. administering to the individual a diagnostically effective amount of a detectably labeled **chemotactic peptide** X--Y--Leu--Phe--[Z].sub.n --W wherein: X is an amino protecting group, Y is ##STR10## where R.sup.1 is benzyl, alkyl, or --CH.sub.2 --CH.sub.2. . . is an intermediary functional group, .nu. is 0 or 1, and M.sup.1 is a diagnostically detectable label; and wherein the **chemotactic peptide** substantially accumulates at the site of infection or **inflammation** and does not substantially accumulate at a site that is not infected or **inflamed**; and b. detecting the **chemotactic peptide**.

7. The method of claim 6 wherein the detectably labeled **chemotactic peptide** is X--Met--Leu--Phe--[Z].sub..nu.

--W.

17. The method of claim 1 wherein the detectably labeled **chemotactic peptide** is selected from the group consisting of N-For-Nle-Leu-Phe-Nle-Tyr-Lys-DTPA N-For-Met-Leu-Phe-Pu-DTPA N-For-Nle-Leu-Phe-Lys-DTPA N-For-Nle-Leu-Phe-Lys(NH.sub.2)-DTPA N-For-Met-Leu-Phe-Lys-DTPA N-For-Met-Leu-Phe-D-Lys (NH.sub.2)-DTPA N-Ac-Nle-Leu-Phe-Lys(NH.sub.2)-DTPA N-Carbobenzoxy-Met-Leu-Phe-Methyl Ester IBOC-L-Methionyl (sulfoxide)-L-Leu-L-Phe-L-lysinamide IBOC-L-Norleucyl-L-Leu-L-Phe-L-lysinamide. . .

38. A method of detecting a site of infection or **inflammation** in an individual which comprises: a. administering to the individual a diagnostically effective amount of a detectably labeled **chemotactic peptide** X--Y--Leu--Phe--[Z].sub.n --W wherein: X is an amino protecting group of the structure ##STR13## where R.sup.3 is selected from the group. . . K is DTPA, EDTA, or HYNIC, .nu. is 1, and M.sup.1 is .sup.111 In or .sup.99m Tc; and wherein the **chemotactic peptide** substantially accumulates at the site of infection or **inflammation** and does not substantially accumulate at a site that is not infected or **inflamed**; and b. detecting the **chemotactic peptide**.

59. A detectably labeled **chemotactic peptide** X--Y--Leu--Phe--[Z].sub.n --W wherein: X is an amino protecting group of the structure ##STR14## where R.sup.3 is selected from the group. . . K is DTPA, EDTA, or HYNIC, .nu. is 1, and M.sup.1 is .sup.111 In or .sup.99m Tc; and wherein the **chemotactic peptide** substantially accumulates at the site of infection or **inflammation** and does not substantially accumulate at a site that is not infected or **inflamed**.

65. A pharmaceutical preparation of the **chemotactic peptide** of claim 59 suitable for parenteral administration.

66. A **chemotactic peptide** selected from the group consisting of N-For-Nle-Leu-Phe-Nle-Tyr-Lys-DTPA, N-For-Met-Leu-Phe-Pu-DTPA, N-For-Nle-Leu-Phe-Lys-DTPA, N-For-Nle-Leu-Phe-Lys(NH.sub.2)-DTPA, N-For-Met-Leu-Phe-Lys-DTPA, N-For-Met-Leu-Phe-D-Lys (NH.sub.2)-DTPA, N-Ac-Nle-Leu-Phe-Lys(NH.sub.2)-DTPA, IBOC-L-Methionyl (sulfoxide)-L-Leu-Phe-L-lysinamide, IBOC-L-Norleucyl-L-Leu-L-Phe-L-lysinamide, N-For-L-Methionyl (sulfoxide)-L-Leu-L-Phe-DTPA-L-Lys, N-For-L-Methionyl. . . Solvated, N-Carbamyl-Met-Leu-Phe-Lys Amide Solvated, N-Trimethylacetyl-Met-Leu-Phe-Lys Amide Solvated, Isobutyloxycarbonyl-Met-Leu-Phe-Lys Amide Solvated, and N-For-Nle-Leu-Phe-Nle-Tyr-N.sup..epsilon. -DTPA-Lys N-Adamantylurea-Met-Leu-Phe N-Cinnamoyl-Met-Leu-Phe P-Tolylurea-Met-Leu-Phe M-Tolylurea-Met-Leu-Phe N-Cinnamoyl-Phe-Leu-Phe-Leu-Phe wherein said **chemotactic peptide** is labeled with a diagnostically detectable label, and said **chemotactic peptide** is capable of accumulating at a site of infection or **inflammation** in an individual and does not substantially accumulate in said site in the absence of infection or **inflammation**.

70. A therapeutic composition comprising a **chemotactic peptide** X--Y--Leu--Phe--[Z].sub.n --[T].sub..alpha. wherein: X is an amino protecting group of the structure ##STR15## where R.sup.3 is selected from the group. . . n is 0 or 1, and T is a therapeutic agent, and .alpha. is 0 or 1; and wherein the **chemotactic peptide** substantially accumulates at the site of infection or **inflammation** and does not substantially accumulate at a site that is not infected or **inflamed**.

L5 ANSWER 4 OF 7 USPATFULL
AN 97:47075 USPATFULL
TI Peptides modified by the phosphine group for marking with 99M TC and 186-188 RE or paramagnetic agents
IN Mazzi, Ulderico, Verona, Italy
Lunghi, Fabio, Moncrivello, Italy
PA Sorin Radiofarmaci SRL, Milan, Italy (non-U.S. corporation)
PI US 5635158 19970603
AI US 95-494105 19950623 (8)
DT Utility
EXNAM Primary Examiner: Hollinden, Gary E.; Assistant Examiner: Hartley, Michael G.
LREP Hoare, Jr., George P.Rogers & Wells
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 505
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . molecule with biological properties useful for diagnosis, radiotherapy or magnetic resonance, and is selected from avidin, biotin, low-density lipoprotein (LDL), **chemotactic peptides** having specificity for **inflammatory** sites and infection processes, peptides having specificity for tumours, peptides having specificity for blood clots and atherosclerotic plaques, cytotoxic agents,. . .

L5 ANSWER 5 OF 7 USPATFULL
AN 95:67219 USPATFULL
TI Chemotactic wound healing peptides
IN Postlethwaite, Arnold E., 635 Bethany Rd., Eads, TN, United States 38028
Seyer, Jerome, 1412 Carr Ave., Memphis, TN, United States 38104
Kang, Andrew, 2334 Massey Rd., Memphis, TN, United States 38119
PI US 5436228 19950725
AI US 93-127909 19930928 (8)
RLI Continuation-in-part of Ser. No. US 90-626631, filed on 12 Dec 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Warden, Jill; Assistant Examiner: Huff, Sheela J.
LREP Scully, Scott, Murphy & Presser
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 1259
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A **chemotactic peptide** having an amino acid sequence as set forth in SEQ ID NO. 1, or a homolog, or an analog thereof.
. . .
2. A **chemotactic peptide** wherein the amino acid sequence of the peptide is selected from the group consisting of SEQ ID NO. 1, SEQ.. . .
3. A **chemotactic peptide** wherein the amino acid sequence of the peptide is selected from the group consisting of SEQ ID NO. 6, SEQ. . .
4. A **chemotactic peptide** wherein the amino acid sequence of the peptide is selected from the group consisting of SEQ ID NO. 11, SEQ. . .
5. A **chemotactic peptide** wherein the amino acid sequence of the peptide is selected from the group consisting of SEQ ID NO. 16, SEQ. . .

6. A method for inducing either in vitro or in vivo chemotaxis of fibroblasts and/or **inflammatory** cells comprising administering to the fibroblasts and/or **inflammatory** cells at least one peptide of any one of claims 1-5 in an amount effective to induce chemotaxis.

7. A method for inducing or a homolog thereof and a pharmaceutically acceptable carrier proliferation of fibroblasts and/or **inflammatory** cells comprising administering to the fibroblasts and/or **inflammatory** cells at least one peptide of any one of claims 1-5 in an amount effective to induce cellular proliferation.

10. A **chemotactic peptide** consisting of a seven amino acid sequence as set forth in SEQ ID NO. 1.

11. A **chemotactic peptide** having an amino acid sequence selected from the group consisting of SEQ ID NO. 1, SEQ ID NO. 2, SEQ. . .

12. An analog of a **chemotactic peptide** set forth in SEQ ID NO. 6 having a cysteine residue attached to the C-terminal end of said peptide.

13. An analog of a **chemotactic peptide** set forth in any one of claims 1-5 having a cysteine residue attached to the C-terminal or N-terminal end of. . .

L5 ANSWER 6 OF 7 USPATFULL
AN 94:112721 USPATFULL
TI Imaging tissue sites of inflammation
IN Morgan, Jr., A. Charles, Edmonds, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 5376356 19941227
AI US 91-726894 19910708 (7)
RLI Continuation of Ser. No. US 89-324285, filed on 14 Mar 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Stoll, Robert L.; Assistant Examiner: Covert, John M.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1580
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. A method of imaging tissue sites of **inflammation** comprising: (1) radiolabeling a recognition agent, wherein said agent is a **chemotactic peptide**, an eosinophilotactic peptide or wherein the agent is capable of interacting selectively with a **chemotactic peptide** receptor associated with activated leukocytes accumulated at said tissue site; (2) infusing radiolabeled recognition agent into a patient; and (3) imaging said tissue sites, whereby medical conditions involving tissue damage mediated by **inflammation** may be detected, evaluated or monitored.

3. A method of claim 1, wherein said **chemotactic peptide** receptor is a receptor for f-met-leu-phe.

4. A method of claim 1, wherein said **chemotactic peptide** is f-met-leu-phe.

5. A method of claim 1, wherein said **chemotactic peptide** is radiolabeled via an additional tyrosine, lysine, cysteine or phenylalanine residue synthesized as part of the peptide.

6. A method of [redacted]im 1, further comprising: (4) [redacted]paring **inflammation** site localization of said **chemotactic peptide** and reticuloendothelial system localization of said peptide, wherein exhibition of a substantial affinity of said peptide for circulating or reticuloendothelial. . . by receptors of non-activated leukocytes, thereby producing a modified peptide capable of preferentially binding to activated leukocytes at sites of **inflammation**.

L5 ANSWER 7 OF 7 USPATFULL
AN 91:6877 USPATFULL
TI Imaging tissue sites of inflammation
IN Morgan, Jr., A. Charles, Edmonds, WA, United States
Anderson, David C., Seattle, WA, United States
PA NeoRx Corporation, United States (U.S. corporation)
PI US 4986979 19910122
AI US 89-364687 19890609 (7)
RLI Continuation-in-part of Ser. No. US 89-324285, filed on 14 Mar 1989
DT Utility
EXNAM Primary Examiner: Maples, John S.
LREP Stoel, Rives
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1617
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A method of imaging a tissue site of **inflammation** comprising: (1) withdrawing leukocytes from a patient; (2) constructing a **chemotactic peptide** or a fragment, a derivative or analog thereof containing an affinity label and a radionuclide label; (3) incubating leukocytes of. . . the labeled peptide and leukocytes resulting from step (4); and (6) imaging said tissue site, whereby medical conditions involving tissue **inflammation** may be detected, evaluated and monitored.

```
s (formyl peptide#)/clm

          3946 FORMYL/CLM
             4 FORMYLS/CLM
          3950 FORMYL/CLM
                 ((FORMYL OR FORMYLS)/CLM)
          6942 PEPTIDE#/CLM
L6           4 (FORMYL PEPTIDE#)/CLM
                 ((FORMYL(W)PEPTIDE#)/CLM)

=> d pn,ti,kwic 1-4


L6   ANSWER 1 OF 4  USPATFULL
PI     US 4851563  19890725
TI     Method for the removal of the formyl group from an ester of an N-formyl
       peptide or N-formyl aminoacid
CLM    What is claimed is:
       1. A method for removing the formyl group from N-formyl
     peptide esters or N-formyl amino acid esters comprising
       contacting said ester with urea in the presence of a polar solvent and.
       . .

L6   ANSWER 2 OF 4  USPATFULL
PI     US 4427660  19840124
TI     Formyl-methionyl chemotatic peptide antibiotic conjugates useful in
       treating infections
CLM    What is claimed is:
       1. An N-formyl peptide-antibiotic complex selected
       from the group consisting of f-Met-Leu-Phe-R, f-Met-Met-Phe-R,
       f-Met-Met-Met-R, f-Nle-Leu-Phe-R, f-Met-Leu-Phe-Lys-R, and their
       pharmaceutically acceptable non-toxic acid addition salts,. . .
       2. The N-formyl peptide-antibiotic complex of claim
       1, wherein R is silver sulfadiazine, which is f-Met-Leu-Phe-silver
       sulfadiazine.

       3. The N-formyl peptide-antibiotic complex of claim
       1, wherein R is silver sulfadiazine, which is f-Met-Met-Phe-silver
       sulfadiazine.

       4. The N-formyl peptide-antibiotic complex of claim
       1, wherein R is silver sulfadiazine, which is f-Met-Met-Met-Met-silver
       sulfadiazine.

       5. The N-formyl peptide-antibiotic complex of claim
       1, wherein R is silver sulfadiazine, which is f-Nle-Leu-Phe-silver
       sulfadiazine.

       6. The N-formyl peptide-antibiotic complex of claim
```

1, wherein R is silver sulfadiazine, which is f-Met-Leu-Phe-Lys-silver sulfadiazine.

7. The N-**formyl peptides** of claim 1 wherein said antifungal agent is miconazole.

8. The N-**formyl peptides** of claim 1 wherein said antifungal agent is clotrimazole.

9. The N-**formyl peptides** of claim 1 wherein said antifungal agent is nystatin.

10. The N-**formyl peptides** of claim 1 wherein said antifungal agent is amphotericin B.

L6 ANSWER 3 OF 4 USPATFULL
PI US 4071511 19780131
TI Method of removing formyl groups from N-formyl-amino acid and N-formyl-peptide esters having free carboxyl groups
CLM What is claimed is:
1. A method of removing the formyl group from the nitrogen atom of an N-formyl-amino acid ester or N-**formyl-peptide** ester having a free carboxyl group which comprises contacting said ester with a strong acid in a mixture of water. . .

L6 ANSWER 4 OF 4 USPATFULL
PI US 4021418 19770503
TI Method of removing formyl groups from N-formyl-amino acid and N-formyl-peptide esters
CLM What is claimed is:
1. A method of removing the formyl group from the nitrogen atom of an N-formyl-amino acid ester or N-**formyl-peptide** ester which comprises reacting said ester with an at least equimolecular amount of hydroxylamine in an inert, liquid medium until. . .

```
(f-Met-Leu-Phe?)

        741568 F
         91851 MET
           112 METS
         91914 MET
                 (MET OR METS)
         11355 LEU
            21 LEUS
           193 LEI
            74 LEIS
         11579 LEU
                 (LEU OR LEUS OR LEI OR LEIS)
        404382 PHE?
L7         118 (F-MET-LEU-PHE?)
                 (F(W)MET(W)LEU(W)PHE?)

=> s l7/clm

        227448 F/CLM
          3229 MET/CLM
             7 METS/CLM
          3231 MET/CLM
                 ((MET OR METS)/CLM)
          2469 LEU/CLM
             2 LEUS/CLM
             5 LEI/CLM
             4 LEIS/CLM
          2479 LEU/CLM
                 ((LEU OR LEUS OR LEI OR LEIS)/CLM)
        112333 PHE?/CLM
L8           5 ((F-MET-LEU-PHE?/CLM))
                 ((F(W)MET(W)LEU(W)PHE?)/CLM)

=> d bib, kwic 1-5


L8  ANSWER 1 OF 5  USPATFULL
AN      95:29712  USPATFULL
TI      Biological mediators of immune functions
IN      Elgebaly, Salwa A., Bloomfield, CT, United States
PA      The University of Connecticut, Farmington, CT, United States (U.S.
        corporation)
PI      US 5403914  19950404
AI      US 92-975640  19921113 (7)
RLI     Continuation-in-part of Ser. No. US 92-852890, filed on 17 Mar 1992, now
        abandoned which is a continuation of Ser. No. US 91-649154, filed on 1
        Feb 1991, now abandoned which is a continuation of Ser. No. US
        87-107280, filed on 9 Oct 1987, now abandoned
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DT Utility
EXNAM Primary Examiner: [redacted]ill, Jr., Robert J.; Assistant [redacted]aminer: Huff, Sheela J.
LREP Chilton, Alix & Van Kirk
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 605
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . level of neutrophil chemotactic activity of the mediator is 5% to 700% the maximum chemotactic response of the synthetic tripeptide **f-Met-Leu-Phe** used as a standard.

L8 ANSWER 2 OF 5 USPATFULL
AN 94:112721 USPATFULL
TI Imaging tissue sites of inflammation
IN Morgan, Jr., A. Charles, Edmonds, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 5376356 19941227
AI US 91-726894 19910708 (7)
RLI Continuation of Ser. No. US 89-324285, filed on 14 Mar 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Stoll, Robert L.; Assistant Examiner: Covert, John M.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1580
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
3. A method of claim 1, wherein said chemotactic peptide receptor is a receptor for **f-met-leu-phe**.

4. A method of claim 1, wherein said chemotactic peptide is **f-met-leu-phe**.

L8 ANSWER 3 OF 5 USPATFULL
AN 94:49073 USPATFULL
TI Diagnostic test procedure for urinary tract inflammatory condition
IN Elgebaly, Salwa A., Bloomfield, CT, United States
PA The University of Connecticut, Farmington, CT, United States (U.S. corporation)
PI US 5318891 19940607
AI US 92-912072 19920708 (7)
RLI Continuation of Ser. No. US 90-521522, filed on 10 May 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Kepplinger, Esther L.; Assistant Examiner: Wortman, Donna C.
LREP Chilton, Alix & Van Kirk
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 323
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
3. The diagnostic test procedure of claim 1 wherein the standard reagent is **f-Met-Leu-Phe**.

4. The diagnostic test procedure of claim 1 including the step of using the tripeptide f[redacted]t-Leu-Phe during the assay as a positive standard and wherein the percent maximum chemotactic response to said fluid is at least. . .

L8 ANSWER 4 OF 5 USPATFULL
AN 87:87564 USPATFULL
TI Chemotactic assay for immunogenicity
IN Palladino, Michael A., San Mateo, CA, United States
PA Genentech, Inc., San Francisco, CA, United States (U.S. corporation)
PI US 4714674 19871222
AI US 85-707005 19850228 (6)
DT Utility
EXNAM Primary Examiner: Marantz, Sidney; Assistant Examiner: Saunders, David A.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 481
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . claim 10 wherein the composition is chemotactic if it exhibits greater than about 10 percent of the chemotactic activity of **f-Met-Leu-Phe**.

L8 ANSWER 5 OF 5 USPATFULL
AN 84:4567 USPATFULL
TI Formyl-methionyl chemotatic peptide antibiotic conjugates useful in treating infections
IN Schiffman, Elliott, Chevy Chase, MD, United States
Altman, Leonard C., Seattle, WA, United States
PA Research Corporation, New York, NY, United States (U.S. corporation)
PI US 4427660 19840124
AI US 82-354357 19820303 (6)
DT Utility
EXNAM Primary Examiner: Phillips, Delbert R.
LREP Scully, Scott, Murphy and Presser
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 941
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. An N-formyl peptide-antibiotic complex selected from the group consisting of **f-Met-Leu-Phe**-R, f-Met-Met-Phe-R, f-Met-Met-Met-R, f-Nle-Leu-Phe-R, **f-Met-Leu-Phe**-Lys-R, and their pharmaceutically acceptable non-toxic acid addition salts, wherein R is an agent selected from the group consisting of sulfonamide. . .
2. The N-formyl peptide-antibiotic complex of claim 1, wherein R is silver sulfadiazine, which is **f-Met-Leu-Phe**-silver sulfadiazine.

6. The N-formyl peptide-antibiotic complex of claim 1, wherein R is silver sulfadiazine, which is **f-Met-Leu-**